The use of intralipid therapy has been gaining traction as a treatment option for an ever expanding range of toxicities. While it has not quite become the standard of care, it has been viable for patients in the veterinary field and has been reported as case studies in the human field. Veterinary literature has reviewed intravenous lipid emulsion therapy (ILE) [1,2] and published case reports or studies are available noting efficacy in toxicities including macrocyclic lactones [3,4], baclofen [5], beta-blockers, calcium channel blockers [6], NSAID [7,8], bromethalin [9], lidocaine [10], permethrin toxicity [11,12], tricyclic antidepressants (13). Intravenous lipid emulsion (ILE) in human literature has been reported as a therapy for local anesthetic [14,15], calcium channel blocker [16,17], psychotropic medication [18], glyphosate-surfactant herbicide toxicities[19] and even cocaine overdosage [20]. Original work performed by Weinberg noted a response in rats with bupivicaine induced systole with lipid emulsion [21]. How exactly ILE works is not certain but two theories are considered. The “lipid sink” theory is most commonly considered the primary mode of action. In this theory, the formation of a lipid compartment within the intravascular space can serve as a “sink” into which the lipophilic drug will be drawn into. The drug is then excreted/metabolized. Determination of a drug's lipophilicity may be noted by its log P value. A value >1 indicates lipophilic compound which may move into the temporary lipid phase and be less distributed throughout the body. The formulation of ILE utilized may play a role and supports the “lipid-sink” theory based on one study [22]. This theory has been supported in two case reports that followed plasma ropivacaine [15] and serum verapamil concentrations [17]. An alternate theory is that the lipid provides an energy source for the cardiac myocytes by increasing the availability of FFA. The increase of FFA may also aid in increasing the activation of voltage-gated calcium channels in the myocardium, increasing cytosolic calcium channels. This mechanism may be most important in cases of calcium-channel blockade [23,24].

There has not been an absolute protocol established for administration of intralipid therapy. A commonly utilized protocol includes an IV bolus of 20% ILE (1.5 mL/kg) followed by a continuous rate infusion of 0.25 mL/kg/min for 30–60 minutes[1]. Intralipid 20% (Baxter) is the most commonly referred to solution. It is composed 20% Soybean Oil, 1.2% Egg Yolk Phospholipids, 2.25% Glycerin, and Water for Injection. It may be infused through a peripheral intravenous catheter (350 mOsmol/kg water) without the use of a filter. The solution must be handled aseptically. Complications of ILE therapy may include fever, hyperlipemia, thrombocytopenia, hemolysis, prolonged coagulation times, seizures or anaphylactoid reactions to the soybean component. In one cat corneal lipidosis was suspected following treatment for ivermectin toxicity [12]. The ph of Intralipid may vary from 6-9 pending where it is is in its shelf-life which should be taken into account in the individual patient. While the log P value predicts the lipophilicity of a drug, other factors such as distribution, patient pH, intravascular volume and oxygenation status affect response to ILE. Restoration of intravascular volume and oxygenation should be corrected prior to initiating ILE treatment. (Sources on rear of page.)
Ask the Vet Sources:


